Effect of Endurance Exercise Training on Left Ventricular Size and Remodeling in Older Adults With Hypertension

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Background. It is not known whether exercise training can induce a reduction of blood pressure (BP) and a regression of left ventricular hypertrophy (LVH) in older hypertensive subjects. This study was designed to determine whether endurance exercise training, by lowering BP, can induce regression of LVH and left ventricular (LV) concentric remodeling in older hypertensive adults.

Methods. We studied 11 older adults with mild to moderate hypertension (BP 152.0 ± 2.5/91.3 ± 1.5 mm Hg, mean ± SE), 65.5 ± 1.2 years old, who exercised for 6.8 ± 3.8 months. Seven sedentary hypertensive (BP 153 ± 3/89 ± 2 mm Hg) subjects, 68.5 ± 1 years old, served as controls. LV size and geometry and function were assessed with the use of two-dimensional echocardiography.

Results. Exercise training increased aerobic power by 16% (p < .001), and it decreased systolic (p < .05) and diastolic (p < .05) BP, LV wall thickness (from 12.8 ± 0.4 mm to 11.3 ± 0.3 mm; p < .05), and the wall thickness-to-radius (h/r) ratio (from 0.48 ± 0.02 to 0.41 ± 0.01; p < .05). There were no significant changes in the controls. The changes in LV mass index (ΔLVMI) were different between the two groups. LV mass index decreased in the exercise group (ΔLVMI = -14.3 ± 3.3 g) but not in the controls (ΔLVMI = 1.4 ± 4.1 g; p = .009). A multiple stepwise regression analysis showed that among clinical and physiological variables including changes in resting systolic BP, aerobic power, body mass index, and systolic BP during submaximal and maximal exercise, only the reduction in resting systolic BP correlated significantly with a regression of concentric remodeling (Δ h/r ratio r = .80; p = .003). The other variables did not add to the ability of the model to predict changes in the h/r ratio.

Conclusions. The data suggest that exercise training can reduce BP and induce partial regression of LVH and LV concentric remodeling in older adults with mild to moderate hypertension.

Left ventricular hypertrophy (LVH) in hypertension is an adaptive process mediated, in part, by a chronic and sustained elevation of systolic blood pressure (BP) and systolic overload. The progressive increase in arterial stiffness with advancing age alters ventricular-arterial coupling in a fashion similar to hypertension and also results in increases in left ventricular (LV) wall thickness and mass (1). Changes in LV structure and geometry in hypertension are characterized by a large increase in LV wall thickness without a significant increase in LV cavity diameter, resulting in increases in the LV wall thickness-to-radius (h/r) ratio (concentric remodeling) and LV mass (concentric LVH). Although concentric LVH and remodeling can be viewed as a useful adaptation that helps maintain stroke volume by preventing an excessive increase in LV systolic wall stress, it is often associated with impaired LV diastolic filling (2,3), diminished coronary vasodilator capacity (4), and a higher incidence of serious ventricular arrhythmias and sudden cardiac death (5,6). Therefore, LVH is generally considered as an independent risk factor with significantly higher cardiac morbidity and mortality in hypertensive patients than in those with similar elevations of BP but without LVH (7). A regression of LVH may ameliorate some of the adverse effects associated with LVH, and it is known to lower the incidence of inducible ventricular fibrillation in experimental animals (8).

Endurance exercise training has been shown to be effective in lowering BP in subjects with mild (grade 1) and moderate (grade 2) hypertension (9–11). However, relatively little is known about the effectiveness of exercise in inducing regression of LVH and concentric remodeling in older subjects in whom the hemodynamic compromise caused by LVH is likely to be more profound because of the superimposed age-associated deterioration in cardiac function (12). Therefore, the aim of this study was to determine whether endurance exercise training can, by lowering BP, induce regression of LVH and concentric remodeling and improve LV function in older adults with mild and moderate hypertension.

Methods

Subjects

We studied 11 older sedentary hypertensive subjects (nine men and two women), 65.2 ± 1.4 years of age (mean ± SE), who participated in a 7-month long program of endurance exercise training. These 11 subjects were among 15 hyper-
tensive individuals who were initially considered for participation in the exercise training program. Three subjects (two men and one woman) were excluded: one man because he was on an oral steroid for an ocular lesion, another man because he was on a diuretic for treatment of hypertension, and one woman because adequate echocardiographic images for analysis were not available. Of the remaining 12 subjects, one man dropped out because of a new onset of congestive heart failure; the other 11 subjects completed the program. We also recruited seven additional sedentary hypertensive subjects (five men and two women), 68.5 ± 1 years old, who served as controls. The control subjects were recruited for the exercise program but could not make the commitment to participate for personal reasons, including frequent travel commitments. Therefore, they were advised to see their personal physicians for follow-up and treatment. Eighteen of the subjects (exercise and sedentary) were selected over a 12-month interval because of their elevated BP from a pool of ~100 older subjects, who were initially evaluated for participation in a program funded by the National Institute on Aging to examine adaptations to exercise in older sedentary adults. The remainder were referred to us.

The duration of hypertension was 4.5 ± 2.7 years in the exercise group and 3.0 ± 1 years in the controls. The selection criteria for all subjects included (a) age between 60 and 72 years; (b) mild (grade 1: BP 140–159/90–99 mm Hg) or moderate (grade 2: BP 160–179/100–109 mm Hg) hypertension (13); (c) no symptoms and no clinical evidence of cardiovascular, cerebrovascular, or renal diseases; and (d) no orthopedic or musculoskeletal problems that might interfere with exercise. None of the subjects were current or previous smokers, and none were taking any antihypertensive medications at the time of enrollment in the study. None of the women were treated with hormone replacement therapy. The study was approved by the Human Studies Committee of Washington University. Written informed consent was obtained from each subject.

Blood Pressure Measurement

Three BP measurements were made 1 week apart. The subjects reported to our research facility in the morning (9–11 a.m.) and rested in the sitting position for 15 min. BP was then taken with the use of a mercury sphygmomanometer, with the subjects’ arms supported at heart level. Three BP recordings were made in each arm during each visit, and the recordings were averaged. This procedure was repeated exactly the same fashion following completion of training in the exercise group and after 7 months in the control group. All posttraining BP evaluations were made at least 16 hours after the last bout of exercise.

Exercise Tests and Determination of Maximal \( O_2 \) Uptake Capacity

After clinical screening (i.e., history, physical examination, and pertinent laboratory tests), each subject had a multistage maximal treadmill test (the Bruce Protocol; 14) to (a) detect the presence of exercise-induced myocardial ischemia or serious arrhythmias and (b) facilitate the design of the protocol for determination of maximal \( O_2 \) uptake capacity (\( VO_2\) max). Approximately 2 weeks later, the subjects had another treadmill test for determination of \( VO_2\) max, as described previously (15). After a 4- to 5-minute warm-up exercise at an intensity equal to 70% of the maximal heart rate, the subjects walked at a constant speed with 2% increases in grade every 2 minutes until exhaustion. During exercise the subjects breathed through a one-way valve, and \( O_2 \) and \( CO_2 \) contents of the expired air were measured continually by using a computer-based system as described previously (15). \( VO_2\) max was defined as either the attainment of a plateau of \( VO_2 \) with increasing exercise intensities or a respiratory exchange ratio above 1.10 (15). \( VO_2\) max was determined before and at the end of the training program in the exercise group. In the control group, the final \( VO_2\) max test could not be determined in three men, one because of a new onset of severe osteoarthritis and the other two because they did not wish to have a repeat exercise testing.

Diet

All subjects (exercisers and controls) were advised not to alter their usual diet. No specific dietary modifications, particularly with respect to restrictions of caloric intake, sodium, potassium, and alcohol consumption, were made. This was verified by obtaining 4-day food records from the exercise group before and in the last 2 weeks of the training program.

Body Composition

Waist circumference was measured at the midpoint between the top of the iliac crest and the bottom of the rib cage. Initial and final measurements were made by the same technician. Because the waist circumference data on three of the control group were not available, they were not analyzed for this group.

Left Ventricular Size and Function

We used two-dimensional (2-D) echocardiography, including 2-D guided M-mode and the transitrdial pulsed Doppler diastolic velocity profile (Hewlett Packard Echocardiograph Model 2000) to evaluate LV size, geometry, systolic performance, and diastolic filling dynamics, as previously described (16). The average number of cardiac cycles used for analysis was 18 ± 1 (range: 7–29 cardiac cycles). The end-diastolic dimension (EDD), end-systolic dimension (ESD), and wall thickness (posterior wall and interventricular septum) were measured, and fractional shortening (FS) and LV mass were calculated by using the standard guidelines and formulas (17,18). Left ventricular end-systolic wall stress \( [\sigma_{es} = (ESP \times r)/2h \times (1 + h/2r)] \) was measured as described by Grossman and colleagues (19). End-systolic pressure (ESP), expressed as grams per centimeter, was estimated as \( (2 \times \text{systolic BP} + \text{diastolic BP})/3 \) with the use of a mercury sphygmomanometer. Diastolic-filling dynamics were evaluated with the use of a pulsed-Doppler transmural diastolic flow-velocity profile. The pulsed-Doppler sample volume was placed in the LV cavity at the tip of the mitral valve leaflets, and the transmural velocities were recorded from the apical four-chamber view. The following variables were evaluated: (a) early peak diastolic flow velocity \( (E) \); (b) late or atrial peak diastolic flow velocity \( (A) \); (c) the ratio of the early-to-late peak LV diastolic flow ve-
locities (E/A); (d) early diastolic deceleration time (Dt); and (e) isovolumetric relaxation time.

Echocardiographic and Doppler images were analyzed in a blinded fashion with respect to (a) the identity of the subject, (b) time (i.e., initial vs final examination), and (c) group (i.e., exercisers vs controls). The intra-observer variability for the measurement of LV posterior wall thickness was 4.7%, the intraventricular septal thickness 4.9%, the end-diastolic diameter 1%, and the end-systolic diameter 0.9%.

Exercise Training Program

Exercise training consisted of a 1-month flexibility program followed by a 7-month endurance exercise training program. In the flexibility exercise program, the flexibility exercises involved all the major joints and muscle groups, and they were deemed necessary to prepare the sedentary older subjects for the endurance exercise training and to reduce the likelihood of incurring injuries and muscle soreness. In endurance exercise training, exercise sessions consisted of a 10-minute warm-up followed by 30 to 50 minutes of endurance exercise, including brisk walking, slow jogging, or cycling. The intensity of the exercise was related to maximal heart rate, and determined by monitoring heart rate during the exercise sessions. Initially, the exercise intensity was adjusted to elicit a heart rate between 60–70% of maximal heart rate. After 3 months, exercise intensity was increased to elicit a heart rate of 70–80% of maximal heart rate. The subjects were required to attend four exercise sessions per week. The exercise prescription was evaluated on a weekly basis, taking into consideration the subjects’ symptoms and their BP responses.

Control Group

The controls were contacted periodically to inquire about their physical activity, symptoms, and BP. Three subjects in this group were treated with antihypertensive medications (hydrochlorothiazide in two subjects and a calcium channel blocker, nifedipine, in one subject) by their personal physicians. The medications were not discontinued during final evaluations.

Statistics

A two-way analysis of variance (ANOVA) followed by pairwise multiple comparison procedures (Tukey test) was used for most of the variables to test the significance of the differences between groups (exercise vs controls) and status (initial vs final) as well as Groups × Status interactions. For some variables (e.g., LV mass), however, we found that the power of this test to detect a significant difference between the two groups was too low to provide reliable and conclusive information. Therefore, we used the test to compare the changes (Δ) from initial to final data between the two groups for those variables where there were substantial numerical differences. Paired t-tests were used in the exercise group to assess changes in VO2 max, and heart rate and BP responses during submaximal exercise before and after training. The data that were not normally distributed were analyzed by using the nonparametric Mann-Whitney Rank Sum Test. A least-squares linear regression analysis was performed to assess the relationships between changes in the physiological variables. Furthermore, we used a stepwise multiple linear regression analysis to identify the variables that contributed significantly to the lowering of BP and regression of concentric remodeling by exercise training. Data are presented as mean ± SE. Statistical significance was accepted as p < .05.

Results

Training Program

The subjects exercised an average of 3.5 ± 0.2 days/week, at an intensity equal to 78.0 ± 2.6% of their maximal heart rate for 6.8 ± 0.4 months (excluding 1.4 ± 0.2 months of flexibility and stretching exercises). The training consisted mostly of walking, walk-jog sequences, and cycling.

Body Composition and Diet

There was a modest weight loss in both groups (Table 1). However, the differences in weight loss between the two groups were not significant (p = .25). The average waist circumference decreased from 102.2 ± 2.5 cm to 99.8 ± 2.5 cm (p = .03) in response to training. The changes in body mass index were not significant (Table 1). Because we were not able to obtain adequate dietary data from control subjects, no reliable comparison between the two groups with respect to dietary factors that could affect BP can be made.

The daily consumption of alcohol (before: 17 ± 6 g; after: 14 ± 7 g; p = .44) and Na+ (before: 3532 ± 552 mg; after: 3582 ± 411 mg; p = .89), and total caloric intake (before: 2551 ± 148 kcal/day; after: 2302 ± 208 kcal/day, where 1 kcal = 4.2 kJ; p = .18) were not significantly different between the trained and untrained states. However, daily K+ intake was lower after training (3826 ± 189 mg vs 3300 ± 321 mg; p = .035).

Maximal O2 Uptake

VO2 max, expressed in liters per minute, increased 16% from 2.04 ± 0.15 l/min to 2.37 ± 0.16 l/min (p = .002) after training. When normalized for body weight, VO2 max increased 17% from 23.1 ± 1.7 (ml/kg)/min to 26.9 ± 1.9 (ml/kg)/min (p < .001). There was no change in VO2 max in those control subjects for whom the data were available (1.74 ± 0.32 l/min vs 1.66 ± 0.25 l/min; n = 4).

Blood Pressure and Heart Rate

The initial systolic BP was similar in the two groups (Table 1). The final systolic BP was significantly lower (p < .05) in the exercise group than in the controls (Table 1). Furthermore, systolic BP decreased significantly in response to training, whereas it did not change in the control group (Table 1). The initial diastolic BP was not different between the two groups (Table 1), and it decreased significantly in both groups (Table 1). The extent of reduction in diastolic BP, however, tended to be greater in the exercise group (p = .07). Exercise training (Table 1) reduced the resting systolic BP by 16.5 ± 2.5 mm Hg (p < .001). ESP decreased from 131.5 ± 2.1 to 117.0 ± 3.0 mm Hg (p < .05) in the exercise group but did not change in the controls (131.7 ± 2.8 vs 128.0 ± 1.4 mm Hg; p = NS). Resting heart rate did not change in either group (Table 1).
A multiple stepwise regression analysis showed that among the following variables—changes in VO2 max, heart rate during submaximal exercise, weight, body mass index, and waist circumference—only the reduction in heart rate during submaximal exercise (p = .012) and the increase in VO2 max (p = .045) correlated significantly with the reduction in systolic BP. The other variables in the model did not add to the ability of the equation to predict the training-induced reduction in systolic BP.

Adaptive Responses During Exercise

Training induced no significant changes in heart rate (155 ± 4 before vs 157 ± 3 b/min after; p = .84) or systolic BP (226.4 ± 7.8 mm Hg before vs 232.7 ± 12.4 mm Hg after; p = 0.9, n = 9) during maximal exercise.

During submaximal exercise at a comparable absolute exercise intensity (VO2; 1.55 ± 0.17 l/min before vs 1.59 ± 0.19 l/min after; p = .7), heart rate was significantly slower in the trained state (119 ± 7 b/min vs 111 ± 8 b/min; p = .04). However, the reduction in systolic BP was not statistically significant (204.2 ± 11.4 mm Hg before vs 188.4 ± 6.5 mm Hg after; p = .18).

Left Ventricular Size and Geometry

LV EDDs and ESDs were larger in the exercise group (Table 1). However, the changes in EDD and ESD from initial to final evaluations were insignificant in both groups (Table 1). The initial values for LV posterior wall thickness did not differ between the two groups (Table 1). However, posterior wall thickness decreased significantly by 12% in the exercise group but did not change in the control group (Table 1). LV septal thickness was initially larger in the exercise group than in the controls (Table 1). It decreased significantly by 11% in the exercise group but did not change in the controls (Table 1). Initial LV mass was greater in the exercise group than in the controls (268.5 ± 16 g vs 226.2 ± 19 g; p = .017). However, there were no differences in LV mass between initial and final evaluations (final values: exercise group, 238.9 ± 14.3 g; controls, 225.4 ± 16.8 g, respectively). LV mass normalized by body surface area did not differ between the two groups. It was lower after training, but the difference between the initial and final values did not attain statistical significance, probably because of small sample size (exercise group: 132.4 ± 6.4 g before; 118.1 ± 5.9 g after; controls: 113.2 ± 7.3 g before; 114.6 ± 5.9 g after). The change in LV mass index from initial to final evaluations, however, was significantly different between the two groups, with the exercise group showing a decrease compared with the control group, which actually exhibited a small increase in LV mass index (exercise group: −14.3 ± 3.3 g vs controls 1.4 ± 4.1 g; p = .009). Both women in the exercise group had an LV mass index suggestive of LVH (>90 g/m2). Among the nine men, eight had an LV mass index greater than 110 g/m2, consistent with LVH. All subjects in both groups had LV concentric remodeling, defined as an LV wall thickness-to-radius (h/r) ratio >0.45 (20). The initial LV h/r ratio was not different between the two groups (Table 1). The h/r ratio decreased markedly in response to training (15%, Table 1) but did not change in the controls (Table 1).

Left Ventricular Systolic Function

There were no significant changes in LV systolic shortening or end-systolic wall stress at rest in either the exercise or control group (Table 1). There was a significant inverse correlation between LV systolic shortening and end-systolic wall stress before (r = 0.86; p < .001) and after (r = 0.78;
add to the ability of the equation to predict the training-induced regression of the concentric remodeling.

Controls
There were no significant differences in the physiological variables between initial and final evaluations (Table 1). The difference in age between the two groups was small and insignificant. In the three subjects who were treated with antihypertensive medications, SBP (154 ± 5 vs 146 ± 2 mm Hg) and posterior LV wall thickness (12.0 ± 0.26 vs 11.5 ± 0.4 mm) were lower during the final evaluation. There were also small changes in diastolic BP (88 ± 2 vs 85 ± 4 mmHg), the h/r ratio (0.49 ± 0.03 vs 0.48 ± 0.04), and LV mass index (111 ± 10 g vs 106 ± 7 g).

**DISCUSSION**

The findings of this study suggest that endurance exercise training that is sufficient to lower SBP can induce partial regression of LV concentric remodeling and LVH in older adults with mild or moderate hypertension, as evidenced by significant reductions in the LV posterior wall and septal thicknesses, the h/r ratio, and LV mass. The absence of changes in LV fractional shortening and end-systolic wall stress at rest suggests that endurance exercise training has no significant effect on baseline LV systolic function in older hypertensive subjects. Because LV function was not evaluated during peak exercise in this study, it is not known whether these older hypertensive subjects would show a greater LV systolic reserve capacity during exercise along with reduction of LVH after training. The absence of changes in LV end-systolic wall stress in response to training despite a lower BP is due to a concomitant reduction in LV wall thickness in the trained state. The lack of a statistically significant increase in LV EDD after training in this study is either because the intensity and the duration of the exercise stimulus were not sufficiently high and long to induce physiologic eccentric hypertrophy superimposed on concentric LVH, or the concurrent resolution of concentric LVH by lowering of BP could have prevented detection of physiologic volume-overload LVH. Intense swim training in the hypertensive rats induces a greater increase in LV mass than that observed in hypertension alone, and unlike the findings of this study, is associated with a small increase in the LV mass-to-volume ratio (21.22).

One of the major adaptations to endurance exercise training is a slower heart rate during submaximal exercise at a given absolute work rate. This adaptation is generally attributed to attenuation of sympathetic activity in the trained state, as reflected in a smaller rise in plasma catecholamine concentrations during submaximal exercise (23). The association between the magnitude of reduction in resting SBP and the slower heart rate during submaximal exercise in the trained state suggests that diminished sympathetic activity may play a role in the BP-lowering effect of endurance exercise. Further studies are needed to elucidate the mechanisms responsible for the reduction of BP by exercise training.

The benefits of exercise and weight loss for the treatment of hypertension have now been documented (10). The results of this study suggest that long-term endurance exercise training may induce a significantly greater decrease in SBP.

**Left Ventricular Filling Dynamics**

The E/A ratio was similar in both groups and did not change significantly in response to training (0.96 ± 0.07 vs 1.12 ± 0.08; p = NS). The changes in the E/A ratio normalized for heart rate, E/A = E/A / (R - R)0.5, were also insignificant (Table 1). The changes in LV isovolumetric relaxation time and early diastolic deceleration time (Dt) were not significant (Table 1).

**Relationships Between Systolic BP and LV Geometry**

When all data (N = 36) were analyzed irrespective of group (exercise and controls) or status (initial and final), we found modest but significant correlations between resting systolic BP (SBP) and the h/r ratio (h/r = 0.13 + 0.003 SBP; r = 0.46; p = .005). The training-induced reduction in resting SBP correlated significantly with the decrease in the LV h/r ratio (Δ h/r = -2.2 + 207 ΔSBP; r = 0.797; p = .003; see Figure 1) and LV wall thickness (ΔPWT = -0.3 + 0.07 ΔSBP; r = 0.69; p = .019). There were no significant (p > .05) correlations between the training-induced changes in LV mass, and SBP during either maximal or submaximal exercise (LV mass: r = 0.2, p = .6; LV wall thickness: r = 0.35, p = .4; the h/r ratio: r = 0.26, p = .54).

A multiple stepwise regression analysis showed that among the clinical and physiological variables that could have plausibly contributed to regression of concentric LV remodeling, that is, the training-induced changes in SBP at rest, VO2max, lean body mass, SBP during maximal and submaximal exercise, only the reduction in resting SBP contributed significantly to the decrease in the h/r ratio (r = 0.88; p = .025). The other variables did not significantly
in the older hypertensive adults than is reported in the younger subjects with hypertension (13). However, because of the small number of subjects and lack of a younger age group, our observation with respect to this adaptation should be interpreted with extreme caution until it is confirmed by a large-scale clinical study. In fact, a recent study reported that short-term (i.e., 8 weeks) exercise training (endurance or resistance exercise) induced a smaller reduction in systolic and diastolic blood pressure in older compared with younger and middle-aged Japanese men and women (24). Gender did not influence the effect of exercise on reduction of BP (24).

There are conflicting reports concerning the effectiveness of endurance exercise training in inducing regression of LVH in subjects with hypertension. A recent study reported regression of LVH in response to exercise training and antihypertensive medications in African American subjects with severe hypertension (25). However, others reported earlier that although endurance exercise training was effective in lowering blood pressure, it did not result in a statistically significant reduction in LV mass in the middle-aged hypertensive subjects (26). The reasons for the discrepancy between these findings are not entirely clear. In the study reported by Baglio and colleagues, there was actually a decrease (9.4%) in LV mass of borderline statistical significance (p = .056) in response to endurance exercise training (26). Therefore, it seems likely that a larger sample size could have resulted in a statistically significant reduction in LV mass in that study. Our findings suggest that in addition to antihypertensive medications (27) and salt restriction (28), endurance exercise training may also be effective in partial resolution of LVH and regression of concentric remodeling in older adults.

Mechanisms underlying regression of LVH and concentric remodeling by exercise training are unknown. It is likely that removal of the excess mechanical stimulus to the left ventricle by the BP-lowering effect of endurance exercise is one mechanism responsible for the regression of LVH and concentric remodeling. This notion is supported by our findings of the reasonably good correlations between the magnitude of the reduction in SBP and the decreases in LV wall thickness and the h/r ratio independent of other variables, and also by in vitro studies documenting that mechanical stretch is the primary inciting stimulus for induction of increased cardiac myocyte protein synthesis and hypertrophy (29–34). However, these observations do not necessarily exclude other potential mechanisms that may play an additional role in reducing LVH by training because, in contrast to cardiac myocytes, cardiac fibroblast proliferation that is responsible for increased collagen synthesis and remodeling is mediated primarily by humoral factors such as transforming growth factor-β1, angiotensin II, norepinephrine, endothelin I, and platelet-derived growth factor, instead of mechanical stretch (35–40). This may explain the absence of a stronger correlation between the training-induced reduction of SBP and regression of LV concentric remodeling, and only partial regression of LVH despite normalization of SBP. Exercise training has been reported to reduce myocardial collagen cross-linking in older rats (41). Therefore, it is plausible to speculate that the observed reduction in LV concentric remodeling in our subjects was associated with decreased myocardial collagen cross-linking in the trained state.

Concentric LVH is often associated with impaired diastolic filling and, occasionally, with diminished systolic performance (2, 42). The Doppler-derived E/A ratio is influenced by multiple factors, including changes in heart rate, left atrial driving pressure, preload, afterload, inotropic state, the pattern and magnitude of LV relaxation, and LV compliance (43), each with different adaptive responses to training. Therefore, it is not surprising that the effect of training on the E/A ratio in the basal resting conditions would be variable. It is, however, possible that the training could have resulted in an improvement in LV filling during exercise in our subjects without altering resting LV diastolic function, as reported recently (44).

The limitations of our study include a small number of patients and lack of randomization. However, because of the documented benefit of lowering BP in reducing cardiovascular risks in hypertensive subjects, it would be unethical nowadays to randomize patients into an untreated control group and withhold treatment for several months. Nevertheless, our results should be considered preliminary and our conclusions may not be extrapolated to all hypertensive patients, particularly those with a severe or moderately severe elevation of BP. Despite the limitations of 2-D echocardiography, the reproducibility and reliability of the measured echocardiographic indexes were reasonably good in this study. Furthermore, the absence of significant changes in BP and echocardiographic variables in the controls supports our findings and conclusions.

In summary, our data suggest that partial regression of concentric LVH and LV remodeling is attainable by endurance exercise training in older adults with mild or moderate hypertension who are vulnerable to the harmful effects of LVH because of the superimposed age-associated reduction in cardiac reserve. Furthermore, our results suggest that reduction of LV mass and concentric remodeling is probably, in part, mediated by the training-induced lowering of SBP and removal of systolic overload.

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